Increased Incidence of Asthma in Children of Smoking Mothers

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ABSTRACT. The relationship between parental smoking and both subsequent development of asthma and subsequent lung function (before age 12) was studied in more than 700 children enrolled before age 5. Children of mothers with 12 or fewer years of education and who smoked 10 or more cigarettes per day were 2.5 times more likely (95% confidence interval 1.42 to 4.59; $P = .0018$) to develop asthma and had 15.7% lower maximal midexpiratory flow ($P < .001$) than children of mothers with the same education level who did not smoke or smoked fewer than 10 cigarettes per day. These relationships were independent of self-reported respiratory symptoms in parents. There was no association between maternal smoking and subsequent incidence of asthma or maximal midexpiratory flow among children of mothers with more than 12 years of education. It is concluded that children of lower socioeconomic status may be at considerable risk of developing asthma if their mothers smoke 10 or more cigarettes per day. It is speculated that recently reported increases in prevalence of childhood asthma may be in part related to the increased prevalence of smoking among less educated women. *Pediatrics* 1992;89:21-26; *asthma, tobacco smoke pollution, maternal education, childhood.*

ABBREVIATIONS. FVC, forced vital capacity; FEV_1, forced expiratory volume in 1 second; $FEF_{25\%-75\%}$, maximal midexpiratory flow.

There is increasing evidence suggesting that parental smoking (especially maternal) may be associated with an increased prevalence of respiratory symptoms in infants and toddlers.[1] In older children of smoking parents, reduced levels of lung function[2] and an increased prevalence of airway hyperresponsiveness[3] and skin test reactivity to aeroallergens[3] have also been reported. However, the possible role of maternal smoking in inducing childhood asthma is controversial and not all studies have observed significant effects (reviewed in Ref. 4). This has been attributed to the small size of the observed effects; reductions in spirometric parameters, for example, are on the order of 1% to 5%, values that have been considered unlikely to lead to clinically serious compromise in lung function.[4] Also, most reports dealing with this issue are from cross-sectional surveys[5] and it has been shown[6] that results of this type of study may be confounded by the concurrent relationships of symptoms in children to other familial factors, particularly a relation of reported children's symptoms to parents' symptoms.

It has been recently observed that prevalence and changes in prevalence of smoking in women are highly dependent on level of education.[7] Differences in smoking habits of subjects with less education vs those with more education may influence the effects of maternal smoking on their children's health. However, socioeconomic level is seldom considered as an effect modifier in studies of parental smoking and respiratory status in children. Authors often report results of multivariable analyses after adjusting for maternal education, paternal occupation, or other indices of socioeconomic level. These procedures are efficient in controlling for confounding but they may also conceal important differences in effect between strata. It is reasonable to assume that the degree of exposure to environmental cigarette smoke may be different for children whose parents have different educational backgrounds.[8] If these differences could be demonstrated, they may at least partially explain the different results obtained in studies of the effects of parental smoking on the respiratory health of their children.

It is also important to consider the fact that most available studies rely either only on parental reports and physicians' diagnoses or only on physiologic measures as outcomes. For the former, interpretation of results may be influenced by the reliability and degree of standardization of the questionnaires used,[5] while for the latter, the clinical significance of small changes in physiologic parameters remains unknown.

The aim of this longitudinal study was to determine the relationship of parental smoking at enrollment (before age 5) to both subsequent incidence of asthma and subsequent lung function in a random sample of children.

SUBJECTS AND METHODS

The subjects of this study are enrollees of the Tucson study of chronic obstructive lung disease.[9] Details of enrollment procedure have been given elsewhere.[9] A random, stratified, cluster sample of white, non-Mexican-American households in Tucson, Arizona, was first studied in 1972 to 1973. During subsequent follow-up surveys (nine had been completed by April 1988), all new members of each selected household were automatically enrolled into the study. The subjects of this study are the 786 subjects who were younger than 5 years of age on enrollment into the study at its inception (survey 1, starting in February 1972) or at any subsequent survey and who had at least one follow-up questionnaire available before age 12. Seventy-five percent of these children were enrolled before age 2. Mean age at the last questionnaire available was 7.4 years for those enrolled before 12 months, 7.7 years for those

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enrolled between the ages of 1 and 3 years, and 8.5 for those enrolled between the ages of 3 and 5 years.

Information about cigarette smoking habits and respiratory symptoms among parents was obtained at the time of enrollment of their children. Questions about smoking focused on each parent separately.

Standardized questionnaires on respiratory symptoms were used to ask parents whether they had recurrent wheeze or chronic cough.[9] Mothers also reported the number of years of formal education, and they were classified in two groups: with (>12 years) or without more than a high school education (≤12 years). We chose this variable as an index of socioeconomic status because previous studies have shown that other more complex indices of socioeconomic level add little to or even obscure the relationships observed between level of education and level of lung function or prevalence of respiratory disease.[10]

The main outcome variable for this study was the development of asthma during follow-up. Children who already had a diagnosis of asthma at enrollment (n = 12) were not included. A question of the general form "Has this child been seen by a doctor for asthma in the past year?" was included in all follow-up surveys. Children were considered to have a new diagnosis of asthma from a certain age (called hereafter "incidence of asthma") if they had not seen a physician for this diagnosis previously and, at that age, they had seen a physician for the disease.

For children aged 6 or older, lung function was measured in all but one survey using standard flow-volume curves.[11] Forced vital capacity (FVC), forced expiratory volume in 1 second (FEV_1), and maximal midexpiratory flow ($FEF_{25-75\%}$) were derived from forced expiratory maneuvers as described earlier.[11] For the purpose of this report, the last lung function test available before age 12 for each subject (n = 316) was used.

Relative risks and their confidence intervals and attributable risks were calculated with the techniques described by Rothman.[12] Pooled point estimation of a uniform effect for stratified cumulative incidence data was assessed both by maximum likelihood and by use of an extension of the Mantel-Haenzel estimators.[12] Analysis of variance and standard *t* tests were used when lung function was the outcome variable.

RESULTS

There were 89 new cases of physician-diagnosed asthma among 774 subjects at risk (cumulative incidence of 11.5%). More than two thirds of these subjects were boys (60/89; $P < .01$). Thirty-five subjects had no information about maternal smoking, and no information about paternal smoking was available for 74 subjects. Mothers and fathers were originally classified as current smokers, ex-smokers, and never-smokers. Since there was no significant difference in any outcome variable between ex-smokers and nerver-smokers (data not shown), these two groups were combined. Mothers who said they were current smokers were classified as light (<10 cigarettes per day) or moderate/heavy (≥10 cigarettes per day) smokers. Only 21 (2.7%) mothers were light smokers, whereas 146 (18.6%) mothers smoked 10 or more cigarettes per day. Also, because results (not shown) for children of mothers who were light smokers were very similar to those of never-smokers (n = 441) and ex-smokers (n = 134), these three groups were combined. Table 1 shows the risk of developing asthma during follow-up by maternal and paternal smoking at enrollment. Children of mothers who smoked 10 or more cigarettes per day were almost 70% more likely to have physician-diagnosed asthma than children of mothers who did not smoke or smoked fewer than 10 cigarettes per day ($P = .015$). Conversely, there was no relationship between paternal smoking and subsequent incidence of asthma.

Children of parents who reported having recurrent wheeze or chronic cough at enrollment of their children (n = 405) were almost 3 times more likely than the remainder of the cohort to have physician-diagnosed asthma (gender-adjusted relative risk = 2.94, 95% confidence interval 1.85 to 4.68; $P < .0001$). Table 2 shows the relationship of mothers' smoking habits to incidence of asthma in their children stratified by gender and by parental symptoms. The risk of developing asthma was still almost 60% higher among children of mothers who smoked 10 or more cigarettes per day after adjusting for gender and parental symptoms (relative risk = 1.59, 95% confidence interval 1.03 to 2.44; $P = .036$). The small number of daughters of nonsmoking mothers with symptoms did not allow for a meaningful comparison within this group. There was no significant difference in the effects of maternal smoking on incidence of asthma between the four groups (boys and girls with and without parental respiratory symptoms, respectively) described in Table 2 (χ^2 for heterogeneity = 3.3; $df = 3$; $P = .6$).

Maternal education had no independent effect on cumulative incidence of asthma (unadjusted relative risk of 1.12 and gender-adjusted relative risk of 0.99 when 310 children whose mothers had ≤12 years of formal education were compared with 428 children of mothers with more than high school education; $P = .9$ and 1.0, respectively). However, mothers with 12 or fewer years of formal education were twice as likely to be current smokers than mothers with higher education (27.7% vs 13.3%, $P < .0001$). Within smokers, however, number of cigarettes smoked daily was not significantly higher in less educated mothers than in mothers with more than 12 years of education (20.6 ± 10.9 vs 20.2 ± 8.7; $P = .8$). Table 3 shows the relative risk of physician-diagnosed asthma by maternal smoking at enrollment and by maternal education. Maternal smoking had different effects on cumulative incidence of asthma depending on the level of maternal education. Among children of less educated mothers, the gender-adjusted risk of developing asthma was 2.55 (95% confidence interval 1.42 to 4.59; $P = .0018$) times higher when the mother smoked 10 cigarettes or more than when the mother smoked fewer than 10 cigarettes or did not smoke. The attributable proportion (ie, the proportion of all cases of asthma occurring among children of less educated mothers that is attributable to maternal smoke exposure) was 30.0%. These relationships persisted after stratifying by any parental respiratory symptoms (adjusted relative risk = 2.25, 95% confidence interval = 1.24 to 4.1; $P = .008$). On the contrary, maternal smoking did not significantly increase the risk of developing asthma among children of mothers with more than 12 years of education (gender-adjusted relative risk = 1.03, 95% confidence interval 0.61 to 1.75; $P = .9$). Paternal smoking did not increase the likelihood of developing asthma either in children of less educated or of more educated mothers.

Daughters of smoking mothers had significantly larger FVC values than daughters of nonsmoking mothers (Table 4; $P = .018$). Children of both genders

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TABLE 1. Relationship Between Mothers' and Fathers' Smoking at Enrollment and Subsequent Development of Asthma in Their Children*

	Boys			Girls			RR Adjusted for Gender (95% CI)
	n	% With Asthma	RR	n	% With Asthma	RR	
Mother							
Smokes ≥ ½ pack/d	80	21.3	1.63	63	12.7	1.78	1.68† (1.10–2.58)
Nonsmoking or < ½ pack/d	315	13.0		281	7.1		
Father							
Smokes ≥ ½ pack/d	102	13.7	0.95	85	9.4	1.35	1.06 (0.67–1.69)
Nonsmoking or < ½ pack/d	269	14.5		244	7.0		

* Information about maternal smoking was not available for 35 children, and 74 subjects had no data on paternal smoking. RR, relative risk; CI, confidence interval.
† $P = .018$.

TABLE 2. Relationship of Mothers' Smoking Habits to Incidence of Asthma in Their Children by Parental Respiratory Symptoms and by Gender*

Parental Symptoms	Maternal Smoking	Boys			Girls		
		n	% With Asthma	RR	n	% With Asthma	RR
Yes	Nonsmoker or < ½ pack/d	180	17.0	1.47	134	10.4	2.07
Yes	≥ ½ pack/d	54	25.9		37	21.6	
No	Nonsmoker or < ½ pack/d	135	6.7	1.80	146	4.1	†
No	≥ ½ pack/d	25	12.0		25	0.0	

* Information about maternal smoking was not available for 35 children. Three additional subjects had no data on parental respiratory symptoms. RR, relative risk. The relative risk (95% confidence interval) of developing asthma by maternal smoking adjusted for gender and parental symptoms was 1.59 (1.03 to 2.44) ($P = .036$).
† Not calculable

TABLE 3. Relationship of Mothers' Smoking Habits to Incidence of Asthma in Their Children by Maternal Education*

Maternal Education	Maternal Smoking	Boys			Girls			RR Adjusted for Gender (95% CI)
		n	% With Asthma	RR	n	% With Asthma	RR	
> 12 y	Nonsmoker or < ½ pack/d	198	14.1	0.86	173	8.7	1.44	1.03 (0.61–1.75)
> 12 y	≥ ½ pack/d	33	12.1		24	12.5		
≤ 12 y	Nonsmoker or < ½ pack/d	117	11.1	2.50	107	4.7	2.71	2.55† (1.42–4.59)
≤ 12 y	≥ ½ pack/d	47	27.7		39	12.8		

* Information about maternal smoking was not available for 35 children. One additional subject had no quantitative data on maternal smoking. RR, relative risk; CI, confidence interval.
† $P = .0018$.

TABLE 4. Lung Function in Children (as Percent Predicted) by Maternal Smoking at Enrollment by Gender*

Lung Function/Maternal Smoking	Boys		Girls	
	Mean ± SD (n)	P	Mean ± SD (n)	P
FVC				
Nonsmoker or < ½ pack/d	100.7 ± 13.2 (145)	.5	98.1 ± 12.7 (111)	.018
≥ ½ pack/d	101.0 ± 13.1 (38)		105.3 ± 13.2 (22)	
FEV_1				
Nonsmoker or < ½ pack/d	100.1 ± 12.8 (145)	.2	98.4 ± 12.7 (111)	.4
≥ ½ pack/d	97.7 ± 11.8 (38)		100.9 ± 13.4 (22)	
FEF_{25-75}				
Nonsmoker or < ½ pack/d	98.6 ± 23.3 (145)	.007	99.1 ± 23.3 (111)	.11
≥ ½ pack/d	88.5 ± 20.0 (38)		90.3 ± 24.4 (22)	

* FVC, forced vital capacity; FEV_1, forced expiratory volume in 1 second; FEF_{25-75}, maximal midexpiratory flow.

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whose mothers smoked 10 or more cigarettes per day had lower values for percent predicted $FEF_{25\%-75\%}$ compared with children of mothers who were nonsmokers or smoked fewer than 10 cigarettes per day, although differences reached statistical significance only for boys. Introduction of any parental respiratory symptoms as a covariate did not affect the relationship between maternal smoking and the lung function parameters described above.

We also studied the relationships described in the previous paragraph and in Table 4 after stratifying by maternal level of education. Compared with children of mothers with more than 12 years of education, children of mothers with 12 or fewer years of formal education had significantly lower mean values for percent predicted FVC (101.4% vs 98.2%; $P = .041$) and percent predicted FEV_1 (100.6% vs 97.2%; $P = .023$) and this effect was independent of maternal of smoking and parental symptoms. Maternal smoking (≥10 cigarettes per day) was associated with significantly higher levels of percent predicted FVC in girls, and this was independent of maternal education (105.9% compared with 98.0% for daughters of nonsmoking mothers; $P = .01$). There were no significant differences in percent predicted FVC between sons smoking and nonsmoking mothers (102.0% vs 100.5%; $P = .09$). The effect of maternal smoking on percent predicted $FEF_{25\%-75\%}$ was highly dependent on maternal level of education. Children of mothers who had 12 or fewer years of formal education and smoked 10 or more cigarettes per day had 15.7% lower mean values for percent predicted $FEF_{25\%-75\%}$ than children of mothers of the same level of education who were nonsmokers or smoked fewer than 10 cigarettes per day (82.8% vs 98.5%, respectively; $P < .005$). This effect was more noticeable and reached statistical significance only in boys (data not shown). Maternal smoking had no effect on percent predicted $FEF_{25\%-75\%}$ values in children of mothers who had at least some education beyond high school (98.5% vs 99.0%, respectively; $P = .9$).

DISCUSSION

In this prospective study we showed that the risk of developing asthma before age 12 was two and a half times higher in children whose mothers smoked 10 or more cigarettes per day at enrollment and had 12 or fewer years of formal education than in children of mothers of the same level of education who were nonsmokers or smoked fewer than 10 cigarettes per day. Maternal smoking had no significant effect on the incidence of asthma among children of more educated mothers. These results were independent of reported respiratory symptoms in parents. Likewise, children of less educated mothers who smoked more than 10 cigarettes per day at enrollment had 15% lower values for percent predicted $FEF_{25\%-75\%}$ (a spirometric parameter that reflects intrathoracic airway function) during follow-up compared with children of mothers of similar level of education who smoked fewer than 10 cigarettes per day or did not smoke. Maternal smoking had no significant effect on percent predicted $FEF_{25\%-75\%}$ among children of mothers with more than 12 years of education.

It is unlikely that our results may be explained by some unknown source of bias. To assess smoking habits, we used a questionnaire that was completed by parents when the child was enrolled into the study (before age 2 in 75% of the cases) and before the child's asthma had been diagnosed. Although this does not totally avoid the possibility of smoking parents' overreporting asthma in their children, it does make biases in smoking reports by parents of symptomatic children more unlikely. We had no information on the exact quitting time for ex-smokers, especially if quitting had occurred before or after the pregnancy with the child enrolled in the study. In addition, results for children of nonsmoking mothers were not different from those of children whose mothers were ex-smokers. For these reasons, we added children of mothers who were ex-smokers when the child was enrolled into the study to those of nonsmoking mothers. If any of these mothers who had quit smoking were actually smoking during the pregnancy or the child's early life, results could be biased but toward no effect.

Our group[6] and others[13] have shown that symptomatic parents may overreport respiratory symptoms in their children. Our present results, however, were independent of this possible source of bias. We chose physician-diagnosed asthma (as reported by parents) as outcome because in our population sample this variable has shown stable and meaningful relationships both with lung function parameters[14] and with indices of allergic sensitization.[15] The parallel effects of maternal smoking on cumulative incidence of asthma and on percent predicted $FEF_{25\%-75\%}$ is additional persuasive evidence against reporting bias as a possible explanation of our findings. Moreover, cumulative incidence of asthma was slightly (albeit not significantly) higher in children of more educated, nonsmoking mothers compared with children of less educated, nonsmoking mothers (Table 3). If anything, underreporting of asthma for children of less educated mothers would bias our estimate of relative risk toward no effect.

Cumulative incidence of asthma in our sample (11.5%) was higher than the frequency of asthma reported in other surveys performed in the United States, such as the Second National Health and Nutrition Examination Survey.[16] Methodologic factors may explain these differences; most reported studies are cross-sectional surveys and their main outcome variable is usually prevalence of active asthma.[16] For the purpose of that type of survey, subjects who may have had asthma in the past but who do not have asthma at the time the survey is conducted are not considered to have asthma. Our longitudinal study, vice versa, considered as having asthma any subject who had a new diagnosis of asthma and had active asthma *at any time* during follow-up. In addition, specific characteristics of our target population such as in-migration patterns[17] may also explain the higher incidence of asthma reported herein.

There are many possible factors which may explain the greater effect of maternal smoking on asthma incidence in children of mothers with 12 or fewer years of formal education. Total cigarette consump-

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tion was not higher in less educated than in more educated mothers and, therefore, does not explain our findings. Crowding and worse housing conditions may increase exposure to sidestream cigarette smoke among children of less educated mothers. It could also be argued that knowledge of the possible ill effects of environmental tobacco smoke may have stimulated mothers with better education to avoid smoking when their children were present in the room. However, the great majority of the subjects of this study were enrolled in the 1970s, when there was probably much less public awareness of the health effects of passive smoking. Increased exposures to aeroallergens as well as nutritional factors may make children of less educated mothers more susceptible to environmental tobacco smoke inhalation. It is relevant to note that children of mothers with 12 or fewer years of formal education had significantly lower values for percent predicted FVC and FEV_1, but not for percent predicted $FEF_{25\%-75\%}$, and this association was independent of maternal smoking and parental respiratory symptoms. This confirms a previous report by Vedal and coworkers,[18] who showed that children of lower socioeconomic status had significantly lower mean levels of FVC and forced expiratory volume in 0.75 second ($FEV_{0.75}$) but not of $FEF_{25\%-75\%}$ than the rest of the population. Lung size may thus differ in children of different socioeconomic backgrounds. However, since FVC and FEV_1 are effort-dependent, differences in expiratory muscle strength (and probably in nutritional status) may also explain these findings. If and how these differences may alter susceptibility to environmental tobacco smoke is unknown.

The mechanisms by which maternal cigarette smoke may increase the risk for developing asthma and induce reductions in lung function in children are unknown. Maternal smoking may affect airway development,[2] and we have previously shown that diminished airway function may be a risk factor for recurrent wheezing during infancy and early childhood.[19] Parental smoking is also associated with an increased prevalence of skin test reactivity to common aeroallergens[3,20] and an enhanced bronchial responsiveness in their children,[3] and these effects may increase the risk for the development of asthma. It was recently suggested[21] that maternal smoking during pregnancy and direct postnatal tobacco smoke inhalation may have different effects on the child's lungs. We did not request information on maternal smoking during and after pregnancy, and it is often difficult to separate these effects, because most mothers do not change their smoking habits after the child's birth.[22] Animal models suggest, however, that smoking during pregnancy may alter elastin deposition and increase compliance of the lungs of the fetus.[23] Abel and co-workers[24] found that lung weight (corrected for body weight) was higher in female rats exposed to nicotine during pregnancy than in controls. Interestingly, we found that, independent of maternal education, daughters of smoking mothers had significantly larger FVC values than daughters of nonsmoking mothers. This finding confirms the report by Vedal and coworkers[18] of higher FVC levels in daughters of smoking mothers. Both a higher lung compliance and an increase in the amount of lung parenchyma may explain these findings. Decrements in $FEF_{25\%-75\%}$ paralleled the increases in asthma incidence and were thus observed only in children of smoking mothers with 12 or fewer years of formal education and were more marked in boys. In contrast, increases in FVC were observed only in girls, regardless of maternal education. This suggests that there may be different mechanisms through which maternal smoking affects lung development. Perhaps intrauterine effects are independent of socioeconomic level and may affect girls more than boys, whereas postnatal smoke exposure is a function of specific living conditions and may affect boys more than girls.[3] Further studies are needed to elucidate these issues.

Our data suggest that children of lower socioeconomic status are at increased risk of developing asthma if their mothers smoke 10 or more cigarettes per day. This finding may have important implications for our understanding of recently described changes in the epidemiology of asthma. Several reports have indicated that prevalence of asthma,[25] hospitalization rates for acute asthma,[26] and asthma mortality[27] in children and young adults increased during the period between 1975 and 1985. Data from the National Center for Health Statistics indicate that increases in morbidity and mortality rates for asthma are particularly important among minorities.[27] A recent study[27] suggested that these differences between ethnic groups are in large part attributable to socioeconomic factors. Studies on the prevalence of smoking in the United States also show that cigarette consumption increased until the mid-1970s and declined steadily since then, but until 1985, smoking initiation rates significantly increased among women with 12 or fewer years of education.[7] Our results are therefore compatible with the hypothesis that the recent increases in prevalence and severity of childhood asthma may be at least in part attributable to an increase in the prevalence of smoking among less educated mothers.

In conclusion, our results suggest that many cases of childhood asthma may be prevented through a sustained effort to discourage smoking initiation and to encourage smoking cessation, particularly among less educated women of childbearing age.

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ANNOUNCEMENT
1992 PEDIATRIC CARDIOLOGY EXAMINATION

The American Board of Pediatrics (ABP) will administer the certifying examination in Pediatric Cardiology on Tuesday, August 11, 1992, in three U.S. cities.

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